



저작자표시-변경금지 2.0 대한민국

이용자는 아래의 조건을 따르는 경우에 한하여 자유롭게

- 이 저작물을 복제, 배포, 전송, 전시, 공연 및 방송할 수 있습니다.
- 이 저작물을 영리 목적으로 이용할 수 있습니다.

다음과 같은 조건을 따라야 합니다:



저작자표시. 귀하는 원저작자를 표시하여야 합니다.



변경금지. 귀하는 이 저작물을 개작, 변형 또는 가공할 수 없습니다.

- 귀하는, 이 저작물의 재이용이나 배포의 경우, 이 저작물에 적용된 이용허락조건을 명확하게 나타내어야 합니다.
- 저작권자로부터 별도의 허가를 받으면 이러한 조건들은 적용되지 않습니다.

저작권법에 따른 이용자의 권리는 위의 내용에 의하여 영향을 받지 않습니다.

이것은 [이용허락규약\(Legal Code\)](#)을 이해하기 쉽게 요약한 것입니다.

[Disclaimer](#)

치의학석사 학위논문

A Longitudinal and Comparative Computed-
tomographic Study on Osteoarthritic Change of
the Temporomandibular Joint between Treatment
and Non-treatment Groups in Young Age
젊은 연령의 측두하악관절 골관절염 환자의
골과괴 변화에 관한 전산화단층촬영을 이용한
치료군과 대조군의 종적 비교 연구

2013 년 2 월

서울대학교 대학원
치의과학과 구강내과·진단학 전공
송 환 희

A Longitudinal and Comparative Computed-
tomographic Study on Osteoarthritic Change of the
Temporomandibular Joint between Treatment and Non-
treatment Groups in Young Age

젊은 연령의 측두하악관절 골관절염 환자의
골파괴 변화에 관한 전산화단층촬영을 이용한
치료군과 대조군의 종적 비교 연구

지도교수 이 정 윤

이 논문을 치의과학석사학위논문으로 제출함

2012년 11월

서울대학교 대학원

치의과학과 구강내과·진단학 전공

송 환 희

송환희의 석사학위논문을 인준함

2012년 12월

위 원 장 _____ (인)

부 위 원 장 _____ (인)

위 원 _____ (인)

ABSTRACT

A Longitudinal and Comparative Computed-tomographic Study on Osteoarthritic Change of the Temporomandibular Joint between Treatment and Non-treatment Groups in Young Age

Hwanhee Song, D.D.S.

*Program in Oral Medicine and Oral Diagnosis, Dept. of Dental
Science, Graduate School Seoul National University
(Directed by Associate Professor **Jeong-Yun Lee**, D.D.S., M.S.D., Ph.D.)*

The aim of this study is to assess the longitudinal change of the temporomandibular joint (TMJ) with osteoarthritis (OA) on computed tomography (CT) comparing treatment group with non-treatment group of Korean temporomandibular disorder (TMD) patients in young age.

CT and clinical records of 44 patients diagnosed as TMJ OA based on the evident destructive condylar bone change of the TMJ on CT were reviewed. Treatment group was consisted of 29 patients taken conservative treatments including instruction of behavioral control, physical therapy and occlusal stabilization splint therapy with periodic follow-up. Non-treatment group was consisted of 15 patients taken neither treatment nor additional follow-up but for

explanation on diagnosis. OA change of the TMJ was evaluated on two sets of CT images taken with 1.5±0.5 years' interval. Clinical information at the time of CT examination including prevalence of self-reported pain, range of maximum and comfortable (painless) mouth opening was also investigated and analyzed.

Total 70 joints (47 joints of treatment group and 23 joints of non-treatment group) were divided into 3 categories of improved, no change and worsened CT groups according to the longitudinal OA change on CT images. Joints of improved CT group were more than a half in both treatment (55.3%) and non-treatment groups (60.9%). The overall extent of OA change (destructive change index, DSTI) showed no significant differences by both treatment and time. However, general aspects of DSTI value change were more favorable in treatment group. In treatment group, the initial DSTI of improved OA was higher (3.32, =more destructive) and that of worsened OA was lower (1.86, =less destructive) significantly. And also range of maximum and comfortable mouth opening of treatment group increased significantly ($p=0.02$ and 0.003) compared to non-treatment group. Prevalence of self-reported pain decreased more in treatment group, not statistically significant, though.

The obtained results are as follows:

1. Improved OA joints were the most in numbers in both treatment and non-treatment groups.
2. The extent of destructive change of TMJ OA was not significantly different by time and treatment.
3. In treatment group, although not statistically significant, general aspects of destructive bone change was more favorable compared to non-treatment group.
4. Analyzed clinical information, the range of mouth opening was significantly increased in treatment group. Prevalence of self-reported

pain was also decreased more in treatment group, not statistically significant.

Although the results of this study may imply that the destruction of hard tissue of TMJ OA has its own disease course and less affected by conventional treatment protocols including occlusal stabilization splint therapy than it is expected, to improve not only destructive osseous change of TMJ OA but also clinical symptoms and jaw functioning, conservative treatments including instruction of behavioral control, physical therapy and occlusal stabilization splint therapy with periodic follow-up should be considered.

**Key words: Temporomandibular joint disorder, Osteoarthritis,
Computed tomogram, Occlusal stabilization splint
therapy**

Student Number: 2010-23759

**A Longitudinal and Comparative Computed-
tomographic Study on Osteoarthritic Change of the
Temporomandibular Joint between Treatment and
Non-treatment Groups in Young Age**

Hwanhee Song, D.D.S.

*Program in Oral Medicine and Oral Diagnosis, Dept. of Dental Science, Graduate
School Seoul National University*

*(Directed by Associate Professor **Jeong-Yun Lee**, D.D.S., M.S.D., Ph.D.)*

-CONTENTS-

- I. INTRODUCTION**
- II. MATERIALS AND METHODS**
- III. RESULTS**
- IV. DISCUSSION**
- V. CONCLUSIONS**
- VI. REFERENCES**
- TABLES**
- FIGURES**
- KOREAN ABSTRACT**

I . INTRODUCTION

Osteoarthritis (OA) or degenerative joint disease (DJD) of the temporomandibular joint (TMJ) is defined as a degenerative condition of the joint characterized by deterioration and abrasion of articular tissue and concomitant remodeling of the underlying subchondral bone (Stegenga et al., 1991; Zarb and Carlsson, 1999). The OA is the result of a pathological imbalance of the degenerative and regenerative tissue responses of the joint under a mechanical load. Under a sustained mechanical load which exceeds the adaptive capacity of the joint, the tissue responses of the joint can be changed to degeneration, resulting in the destruction of hard tissue and secondary inflammatory changes (Milam, 2005; Tanaka et al., 2008). The clinical signs and symptoms of OA are usually reported as pain, joint noise, and dysfunctions such as restriction of mouth opening, although not all of them are symptomatic (Onder et al., 2009), which are not different from those of other forms of TMD.

Treatment is usually focused on relief of the symptoms and preventing further progression of degeneration, which includes rest, avoidance of trauma, physical therapy, medication, occlusal splint therapy and surgical interventions if conservative therapy failed to relieve OA (Swintak et al., 1976). A number of study have been suggested the efficacy of these treatments modality on improving clinical signs and symptoms such as decrease pain on functioning and increase of mouth opening with some reliable evidences (Guarda-Nardini et al., 2009; Machon et al., 2010; Manfredini et al., 2009; Mejersjo and Wenneberg, 2008; Onder et al., 2009).

However, it should be noted that diagnostic disagreement between clinical and radiographic findings of TMJ OA have been well described in many studies (Bernhardt et al., 2007; Emshoff and Rudisch, 2001; Look et al., 2010; Ohlmann et

al., 2006; Sato et al., 1996; Schiffman et al., 2010). Based on this information, it cannot be certain that the alleviation of the clinical signs and symptoms implies the end of osseous destruction. Despite that knowledge about the bone changes of TMJ OA, such as flattening, sclerosis, osteophyte, erosion and subchondral bone cyst, is fundamental for correct diagnosis of the disease and adequate treatment planning (Hatcher and Aboudara, 2004), there are few studies investigating neither longitudinal radiographic degenerative bone change nor treatment efficacy toward the bone change.

Meanwhile computed tomography (CT) allows the TMJ to be clearly visualized without the interference of nearby anatomical structures and thus has been regarded as a method of choice to verify cortical bone change of TMJ OA (Kurita et al., 2005). However, there are also few studies on the prevalence and prognosis of osseous changes in TMJ, particularly using CT.

In this study we intended to know how the destructive bone change of TMJ OA progress and whether conservative treatments work toward its osseous change or not. This study reports the results of a 1.5±0.5 years' follow up observation of TMJ OA patients using CT between treatment and non-treatment groups.

II. MATERIALS AND METHODS

1. Subjects

The patients who had attended TMJ and Orofacial Pain Clinic of Seoul National University Dental Hospital complaining of TMD symptoms such as preauricular pain on functioning, noise and restriction of mouth opening etc. from August of 2008 to July of 2010 and whose TMJ condyle have destructive bone change confirmed on CT were reviewed. Among them, the subjects who had taken neither

treatment nor additional follow-up but for explanation on the disease state were selected. These 15 patients of non-treatment group (3 males and 12 females, aged in 20.0 ± 6.3 and 18.3 ± 7.1 years, respectively) were recalled and took the second CT with 1.6 ± 0.6 years' interval. In case of treatment group, conservative treatments include instruction of behavior control, physical therapy, medication and occlusal stabilization splint with periodic follow up. Considering the distribution of age and sex of non-treatment group, we selected 29 patients (6 males and 23 females, aged in 20.5 ± 4 and 18.5 ± 6.1 years, respectively) of treatment group and verified the homogeneity of both groups statistically. CT interval of treatment group was 1.4 ± 0.6 years. The prognosis of the TMJ OA was evaluated by two sets of CT images (treatment group vs. non-treatment group) taken with average of 1.5 ± 0.5 years' interval. This study was approved by the Institutional Research Board of Seoul National University Dental Hospital. (#CRI11009)

2. Acquisition of CT images

CT imagery was acquired in terms of SOMATOM Sensation 10 (Siemens, Munich, Germany) with 0.75mm slice collimation. Patients were positioned in supine and corrected sagittal, corrected coronal, and axial images of the TMJs were reconstructed along the true axes of the mandibular condyle at a slice thickness of 1-2mm.

3. Diagnosis of TMJ OA

TMJ OA diagnosis in the clinic was based on clinical signs and symptoms including typical osteoarthritic changes of the articular surfaces on CT images such as flattening, erosion, sclerosis, osteophyte, subchondral bone cyst and loose joint body, as described previously: articular surface flattening, a loss of the rounded contour of the surface; subcortical sclerosis, any increased thickness of the cortical plate in the load-bearing areas relative to the adjacent non-load bearing areas;

subchondral bone cyst, a cavity below the articular surface that deviates from the normal marrow pattern; surface erosion, loss of continuity of the articular cortex; osteophyte, marginal hypertrophy with sclerotic borders and an exophytic angular formation of osseous tissue arising from the surface; generalized sclerosis, no clear trabecular orientation with no delineation between the cortical layer and trabecular bone that extends throughout the condylar head; loose joint body, a well-defined calcified structure(s) that is not continuous with the disc or osseous structures of the joint (Winocur et al., 2010).

In this study, the joints of which surfaces have erosion and subchondral cyst on CT images were only regarded as destructive change of TMJ OA. Flattening and sclerosis, which were not accompanied by erosion, subcortical cyst, were not regarded as types of destructive change caused by OA. Because it has not been in consensus yet whether the osseous change with low destructivity such as flattening, sclerosis, and osteophyte with stable integrity of cortical line should be diagnosed as OA or not (Hussain et al., 2008).

To determine the extent of the destructive OA change, the condylar surface was divided into three sections in the antero-posterior direction between the apex of the articular eminence and the squamotympanic fissure on the corrected sagittal views and three sections again in the medio-lateral direction between the squamotympanic fissure and the lateral rim of the articular fossa on the corrected coronal views, resulting in nine imaginary sections on the entire condylar surface (Figure 1) (Lee et al., 2011). The number of sections in which erosion, subchondral cyst was observed was counted by the examiner (Song) to represent the extent of the destructive change by the disease, which was named Destructive change Index (DSTI).

4. Comparison of the longitudinal bone change of TMJ OA

The longitudinal bone change by TMJ OA was evaluated based on the comprehensive change of the integrity of the cortical line, the subcortical marrow pattern, and the morphology of the condyle in comparisons of the first and the second CT image sets. The joints were classified into the following three groups by a single examiner (Song): no change group, no visible change of the shape of osseous structures nor the integrity and quality of the cortical line and subcortical marrow pattern; worsened CT group, an increase in the extent or severity of the OA change observed on the first CT images, or the occurrence of any type of additional OA change not observed in the first CT images; improved CT group, a decrease in the extent or severity of the OA change observed in the first CT images with an improvement of the integrity and quality of the cortical line or subcortical marrow pattern. The examiner was blind to any other information about the patient, apart from the CT images. Example images of the three groups are presented in Figure 3 (Lee et al., 2011).

The longitudinal bone change of TMJ OA was evaluated three times with two-week's interval by a single examiner (Song), trained TMJ specialist. Each reading was blinded to the clinical and other radiological diagnosis. In order to verify reliability of readings, the three readings were analyzed by Cohen's kappa value which was 0.739 ($p < 0.005$ in all comparison among three times' readings), substantial reliability. The final decision was made by majority rule based on the results of the three readings.

5. Statistical analysis

Statistical analysis was finally done with 70 joints of 44 patients, excluding 18 joints which were determined to be normal in both of the first and second CT image sets. Homogeneity of both groups as to the distribution of age, sex and CT intervals was verified with T-test. Frequency analysis of the longitudinal bone change of TMJ OA on CT was analyzed by Chi-square tests. Changes of the mouth

opening range, prevalence of self-reported pain and the number of sections with destructive changes (Destructive change Index) were analyzed by repeated-measures ANOVA.

III. RESULTS

1. The composition of treatment and non-treatment groups

The records of 29 patients for treatment group (male 6 and female 25, mean age was 19.0 ± 5.8 years) and 15 patients for non-treatment group (male 3 and female 12, 18.2 ± 6.7 years) were selected. The joints diagnosed with destructive change confirmed on CT images were 70 joints, 47 joints of treatment group and 23 joints of non-treatment group. Age and sex distribution and the time interval of CT examinations between treatment and non-treatment groups were not statistically different by T-test.

2. The longitudinal bone change of TMJ OA on CT

In treatment group, 26 out of 47 joints (55.3%) were classified into the improved CT group, 18 out of 47 (38.3%) into the worsened CT group, and 3 out of 47 (6.4%) into no change group. In non-treatment group, 14 out of 23 joints (60.9%) were classified into the improved CT group, 3 out of 23 (13.0%) into the worsened CT group, and 6 out of 23 (26.1%) into no change group (Table 1). While the distribution of the prognostic three groups turned out to be statistically different between treatment and non-treatment groups, it is noticeable that more than a half of the joints were improved in both groups within 1.5 ± 0.5 years.

3. The extent of destructive change of TMJ OA

DSTI representing the extent of destructive change of TMJ OA was examined on the first (DSTI1) and second (DSTI2) set of CT images for the comprehensive decision of OA changes. The change of DSTI is shown in Table 2, Figure 3 and 4. DSTI changed significantly neither by time nor treatment in the total joint samples by repeated measures ANOVA (Figure 3a). However, DSTI decreased in improved group ($p=0.01$) and increased in worsened group ($p=0.02$) significantly by time (Figure 3b and 3d).

Meanwhile in treatment group, the initial DSTI (DSTI1) of improved OA was higher (3.32, =more destructive) and that of worsened OA was lower (1.86, =less destructive) significantly. DSTI1 value of treatment group showed significant difference ($p=.0003$) between improved and worsened OA group, not DSTI2 value (Figure 4b). Similarly, in control group, DSTI2 value by its prognosis showed significant difference ($p=.018$), not DSTI1 value (Figure 4c). Furthermore, nevertheless not significant, within improved OA, degree of DSTI decrease was larger in treatment group than control group (Figure 3b) and within worsened OA degree of DSTI increase was larger in control group (Figure 3d).

4. Analysis of clinical information

Range of maximum mouth opening (MMO) increased from $44.98\pm 8.83\text{mm}$ to $50.17\pm 5.93\text{mm}$ in treatment group and from $45.26\pm 5.68\text{mm}$ to $46.17\pm 3.58\text{mm}$ in non-treatment group. Range of comfortable (painless) mouth opening (CMO) also increased from $41.94\pm 9.22\text{mm}$ to $49.79\pm 5.96\text{mm}$ in treatment group and from $43.61\pm 6.73\text{mm}$ to $44.74\pm 5.22\text{mm}$ in non-treatment group. Both MMO and CMO increased more significantly in treatment group than non-treatment group ($p=.02$ and $.003$).

Prevalence of self-reported pain on the initial visit was 51.1% of treatment group and 34.8% of non-treatment group, the difference, however, was not statistically significant ($p=.152$). On the secondary CT taking, prevalence of self-reported pain was 8.5% of treatment group and 13.0% of non-treatment group. The prevalence decreased from 51.1% to 8.5% in treatment group and from 34.8% to 13.0% in non-treatment group. Though the prevalence decreased more in treatment group, which was not statistically significant. Other clinical information including Angle's classification, medication and pain on masticatory muscle palpation were also not different significantly between groups.

IV. DISCUSSION

In this study we investigated how the prognosis of destructive osseous change of TMJ OA goes on and is affected by conservative treatments including occlusal stabilization splint in terms of CT examinations repeated by average 1.5 years' interval. Actually very a few studies reported about the disease progress and treatment efficacy on the destructive osseous change of TMJ OA with reliable evidences. In previous studies they investigated treatment modality focused on clinical information only or lacked control group, if not, based on only radiography such as orthopantomogram or transcranial projection view which has limitation of superimposition. As far as authors' knowledge, this study is the first attempt to investigate the longitudinal osseous change of TMJ OA on CT images comparing treatment group with non-treatment group.

Above all, methodologically, statistical homogeneity of both groups, especially distribution of age and sex, had to be verified necessarily, because several studies have reported that age and sex showed a significant association with presence of

bone changes of TMJ (Alexiou et al., 2009; dos Anjos Pontual et al., 2012; LeResche et al., 1991). According to Alexious et al, patients in older age groups are expected to have more frequent and severe bone changes than those in younger age groups (Alexiou et al., 2009). LeResche et al. reported that osteoarthritis of TMJ is more frequent in women (LeResche et al., 1991). For this reason we matched the distribution of age and sex between both groups and restricted all subjects' age under 30 years old. Furthermore baseline osseous state of both groups was not statistically different.

As a result, prognosis of destructive change with TMJ OA improved in more than 50% in about 1.5 years irrespective of treatments. The extent of destructive osseous change of the condyle did not change significantly by time and treatment within total sample. Meanwhile higher DSTI at first was likely to improve later and vice versa in both treatment and non-treatment group. It may be suggested that progressed destructive change of the condyle tends to be stable, while incipient osseous change might be deteriorate further. It can be postulated that once destructive change initiate, it would go its own courses till the joint reaches to a state of musculoskeletally stable relation.

This result may be also in line with some previous study indicating that a radiographically stable terminal stage is reached eventually (de Leeuw et al., 1995; Rasmussen, 1980; Toller and Wilcox, 1978). De Leeuw et al. reported radiographically stable end stage of TMJ OA may be reached within a few years in most cases (de Leeuw et al., 1994). Kurita et al. reported that most of the destructive changes were arrested or slowed in the patients whose symptoms and signs were successfully resolved or reduced (Kurita et al., 2005). According to Toller et al, ongoing radiologic changes took place within 1-3 years (Toller and Wilcox, 1978).

However, these findings should be carefully interpreted not to jump to the improper conclusion that TMJ OA needs not to be treated because it would stop of

itself. Because the extent of destructive osseous change of treatment group was more likely to improve within improved CT group and less likely to be worsened within worsened CT group than that of non-treatment group, without statistical significance though. In addition according to the results of this study, clinical signs and symptoms improved more in treatment group significantly. In treatment group, range of mouth opening was improved more significantly and the prevalence of self-reported pain decreased more not significantly though compared to non-treatment group. It implies that the restoring function may be more favorable in treatment group than non-treatment group.

These also correspond the results of other studies assessed effectiveness of treatments including occlusal splint therapy to reduce clinical TMD signs and symptoms (Brown and Gaudet, 1994; Ekberg et al., 1998; Garefis et al., 1994; Kuttilla et al., 2002; Lee et al., 2011). According to Kuttilla et al. in a controlled study on the effects of occlusal splint therapy in individuals with severe TMJ osteoarthritis, a reduction of clinical signs was seen (Kuttilla et al., 2002). Occlusal splint has been regarded to be beneficial to relieve pain and restore function of not only osteoarthritis but also other subtypes of TMD. However it has been well described about a disagreement between clinical information and radiographic findings of TMJ OA (Bernhardt et al., 2007; Emshoff and Rudisch, 2001; Look et al., 2010; Ohlmann et al., 2006; Sato et al., 1996; Schiffman et al., 2010). Lee et al. reported that the prognosis of OA changes may be independent of the clinical signs and symptoms with 1 years' follow-up and the number of destructive sections did not change with time (Lee et al., 2011). Therefore as to the treatment of TMJ OA, it is necessary to consider the patients' symptom (pain), jaw functioning and bone changes comprehensively.

In addition it is not negligible that on diagnosis all subjects had consultation about TMJ OA and got to know what the problem with their joint is and what to do

for the joints. It is natural that most of them try to protect their joints, even if they cannot afford get outpatient treatments, which might be implicated in these results.

As far as the treatment protocol is conservative and not harmful to the patient, the conventional treatment such as behavioral control, physical therapy and occlusal splint therapy with periodic check up should not be discouraged for the reduction of overloading to the joint. It is logical that the treatment of TMJ OA should be directed to help the joint can reach to a musculoskeletally stable state more easily and quicker, rather than toward the osseous change itself. It seems apparent that to support this inference advanced further study based on more scientific and precise evidences with larger subjects and for longer period would be necessary.

IV. Conclusions

1. Improved OA joints were the most in numbers in both treatment and non-treatment groups.
2. The extent of destructive change of TMJ OA was not significantly different by time and treatment.
3. In treatment group, although not statistically significant, general aspects of destructive bone change was more favorable in treatment group than in non-treatment group.
4. Analyzed clinical information, range of mouth opening was significantly increased in treatment group. Prevalence of self-reported pain was also decreased more in treatment group, not statistically significant.

Although the results of this study may imply that the destruction of hard tissue of TMJ OA has its own disease course and less affected by conventional treatment protocol including splint therapy than it is expected, to improve not only destructive osseous change of TMJ OA but also clinical symptoms and jaw functioning, conservative treatments including instruction of behavioral control, physical therapy and occlusal stabilization splint therapy with periodic follow-up should be considered.

V. REFERENCES

Alexiou K, Stamatakis H, Tsiklakis K (2009). Evaluation of the severity of temporomandibular joint osteoarthritic changes related to age using cone beam computed tomography. *Dento maxillo facial radiology* 38(3):141-147.

Bernhardt O, Biffar R, Kocher T, Meyer G (2007). Prevalence and clinical signs of degenerative temporomandibular joint changes validated by magnetic resonance imaging in a non-patient group. *Ann Anat* 189(4):342-346.

Brown DT, Gaudet EL, Jr. (1994). Outcome measurement for treated and untreated TMD patients using the TMJ scale. *Cranio : the journal of craniomandibular practice* 12(4):216-222.

de Leeuw R, Boering G, Stegenga B, de Bont LG (1994). Clinical signs of TMJ osteoarthritis and internal derangement 30 years after nonsurgical treatment. *Journal of orofacial pain* 8(1):18-24.

de Leeuw R, Boering G, Stegenga B, de Bont LG (1995). Radiographic signs of temporomandibular joint osteoarthritis and internal derangement 30 years after nonsurgical treatment. *Oral surgery, oral medicine, oral pathology, oral radiology, and endodontics* 79(3):382-392.

dos Anjos Pontual ML, Freire JS, Barbosa JM, Frazao MA, dos Anjos Pontual A (2012). Evaluation of bone changes in the temporomandibular joint using cone

beam CT. *Dento maxillo facial radiology* 41(1):24-29.

Ekberg EC, Vallon D, Nilner M (1998). Occlusal appliance therapy in patients with temporomandibular disorders. A double-blind controlled study in a short-term perspective. *Acta odontologica Scandinavica* 56(2):122-128.

Emshoff R, Rudisch A (2001). Validity of clinical diagnostic criteria for temporomandibular disorders: clinical versus magnetic resonance imaging diagnosis of temporomandibular joint internal derangement and osteoarthritis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 91(1):50-55.

Garefis P, Grigoriadou E, Zarifi A, Koidis PT (1994). Effectiveness of conservative treatment for craniomandibular disorders: a 2-year longitudinal study. *Journal of orofacial pain* 8(3):309-314.

Guarda-Nardini L, Manfredini D, Stifano M, Staffieri A, Marioni G (2009). Intra-articular injection of hyaluronic acid for temporomandibular joint osteoarthritis in elderly patients. *Stomatologija / issued by public institution "Odontologijos studija" [et al]* 11(2):60-65.

Hatcher DC, Aboudara CL (2004). Diagnosis goes digital. *American journal of orthodontics and dentofacial orthopedics : official publication of the American Association of Orthodontists, its constituent societies, and the American Board of Orthodontics* 125(4):512-515.

Hussain AM, Packota G, Major PW, Flores-Mir C (2008). Role of different imaging modalities in assessment of temporomandibular joint erosions and osteophytes: a systematic review. *Dento maxillo facial radiology* 37(2):63-71.

Kurita H, Uehara S, Sakai H, Kamata T, Kurashina K (2005). Radiographic follow-up of diseased temporomandibular joints. *Oral surgery, oral medicine, oral pathology, oral radiology, and endodontics* 100(4):427-432.

Kuttila M, Le Bell Y, Savolainen-Niemi E, Kuttila S, Alanen P (2002). Efficiency of occlusal appliance therapy in secondary otalgia and temporomandibular disorders. *Acta odontologica Scandinavica* 60(4):248-254.

Lee JY, Kim DJ, Lee SG, Chung JW (2011). A longitudinal study on the osteoarthritic change of the temporomandibular joint based on 1-year follow-up

computed tomography. *Journal of cranio-maxillo-facial surgery : official publication of the European Association for Cranio-Maxillo-Facial Surgery*.

LeResche L, Dworkin SF, Sommers EE, Truelove EL (1991). An epidemiologic evaluation of two diagnostic classification schemes for temporomandibular disorders. *The Journal of prosthetic dentistry* 65(1):131-137.

Look JO, Schiffman EL, Truelove EL, Ahmad M (2010). Reliability and validity of axis I of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) with proposed revisions. *J Oral Rehabil* 37(10):744-759.

Machon V, Hirjak D, Lukas J (2010). Therapy of the osteoarthritis of the temporomandibular joint. *J Craniomaxillofac Surg*.

Manfredini D, Bonnini S, Arboretti R, Guarda-Nardini L (2009). Temporomandibular joint osteoarthritis: an open label trial of 76 patients treated with arthrocentesis plus hyaluronic acid injections. *Int J Oral Maxillofac Surg* 38(8):827-834.

Mejersjo C, Wenneberg B (2008). Diclofenac sodium and occlusal splint therapy in TMJ osteoarthritis: a randomized controlled trial. *J Oral Rehabil* 35(10):729-738.

Milam SB (2005). Pathogenesis of degenerative temporomandibular joint arthritides. *Odontology / the Society of the Nippon Dental University* 93(1):7-15.

Ohlmann B, Rammelsberg P, Henschel V, Kress B, Gabbert O, Schmitter M (2006). Prediction of TMJ arthralgia according to clinical diagnosis and MRI findings. *Int J Prosthodont* 19(4):333-338.

Onder ME, Tuz HH, Kocyigit D, Kisnisci RS (2009). Long-term results of arthrocentesis in degenerative temporomandibular disorders. *Oral surgery, oral medicine, oral pathology, oral radiology, and endodontics* 107(1):e1-5.

Rasmussen OC (1980). Longitudinal study of transpharyngeal radiography in temporomandibular arthropathy. *Scandinavian journal of dental research* 88(3):257-268.

Sato H, Osterberg T, Ahlqwist M, Carlsson GE, Grondahl HG, Rubinstein B (1996).

Association between radiographic findings in the mandibular condyle and temporomandibular dysfunction in an elderly population. *Acta Odontol Scand* 54(6):384-390.

Schiffman EL, Ohrbach R, Truelove EL, Tai F, Anderson GC, Pan W *et al.* (2010). The Research Diagnostic Criteria for Temporomandibular Disorders. V: methods used to establish and validate revised Axis I diagnostic algorithms. *J Orofac Pain* 24(1):63-78.

Stegenga B, de Bont LG, Boering G, van Willigen JD (1991). Tissue responses to degenerative changes in the temporomandibular joint: a review. *Journal of oral and maxillofacial surgery : official journal of the American Association of Oral and Maxillofacial Surgeons* 49(10):1079-1088.

Swintak EF, Schriver WR, Tsagaris GJ (1976). Symptomatic degenerative joint disease. Report of a case. *Oral surgery, oral medicine, and oral pathology* 42(6):722-725.

Tanaka E, Detamore MS, Mercuri LG (2008). Degenerative disorders of the temporomandibular joint: etiology, diagnosis, and treatment. *Journal of dental research* 87(4):296-307.

Toller PA, Wilcox JH (1978). Ultrastructure of the articular surface of the condyle in temporomandibular arthropathy. *Oral surgery, oral medicine, and oral pathology* 45(2):232-245.

Winocur E, Reiter S, Krichmer M, Kaffe I (2010). Classifying degenerative joint disease by the RDC/TMD and by panoramic imaging: a retrospective analysis. *J Oral Rehabil* 37(3):171-177.

Zarb GA, Carlsson GE (1999). Temporomandibular disorders: osteoarthritis. *Journal of orofacial pain* 13(4):295-306.

Table 1. Frequency analysis of the prognosis of TMJ OA between treatment and non-treatment groups

Prognosis	Treatment group	Non-treatment group	Sum
	N (%)	N (%)	
Improved	26 (55.3)	14 (60.9)	40 (57.1)
No change	3 (6.4)	6 (26.1)	9 (12.9)
Worsened	18 (38.3)	3 (13.0)	21 (30.0)

Table 2. Mean value of the Destructive change Index (DSTI) of treatment group, non-treatment group and total

	Total		Treatment group		Non-treatment group	
	DSTI1	DSTI2	DSTI1	DSTI2	DSTI1	DSTI2
Improved	3.32	1.61	3.77	1.69	2.87	1.53
No-change	2.75	2.08	2.33	1.33	3.17	2.83
Worsened	1.86	3.81	1.39	2.94	2.33	4.67
mean	2.64	2.50	2.50	1.99	2.79	3.01

DSTI, Destructive change Index; DSTI1, DSTI on the first CT; DSTI2, DSTI on the second CT

Figure Legends

Figure 1. Nine imaginary sections of the articular surface of the mandibular condyle

A: The condylar surface was divided into nine imaginary sections in the CT reading

B: The condylar surface was divided into three sections in the antero-posterior direction between the apex of the eminence and the squamotympanic fissure

C: The condylar surface was divided into three sections in the medio-lateral direction between the squamotympanic fissure and the lateral rim of the articular fossa

Figure 2. Examples of the three prognosis types of TMJ OA

A: No change; B: Improved; C: Worsened

The left side in each example is the image of the first CT examination, and the right side is the one of the second.

Figure 3. Changes of the Destructive change index (DSTI) by prognosis

Figure 4. Changes of the Destructive change index (DSTI) by treatment

Figure 5. Changes of the range of maximum and comfortable mouth opening

CMO, Comfortable (painless) Mouth Opening; MMO, Maximum Mouth Opening

Figure 6. Changes of the prevalence of self-reported pain

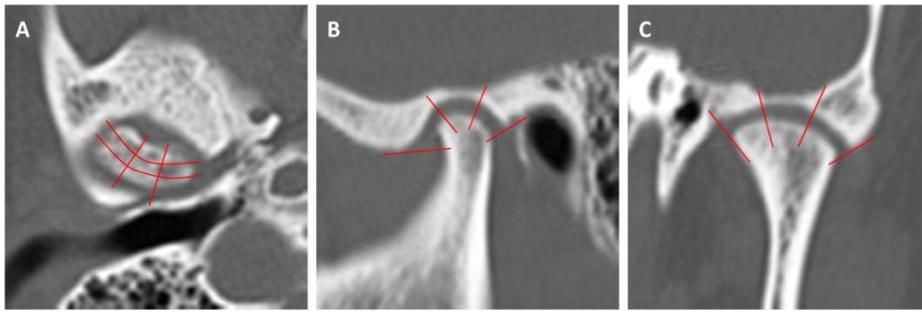


Figure 1.

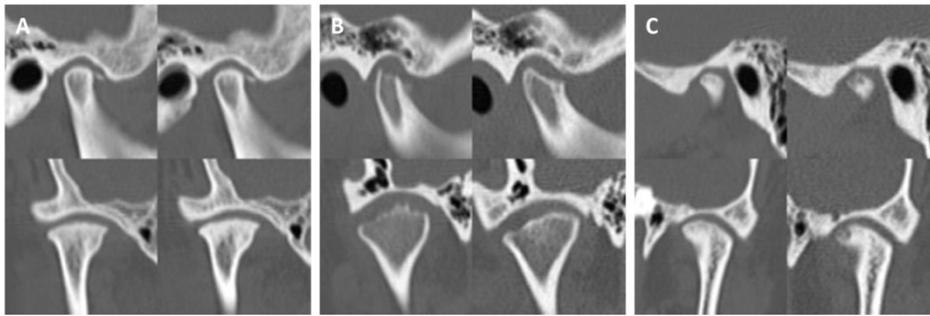


Figure 2.

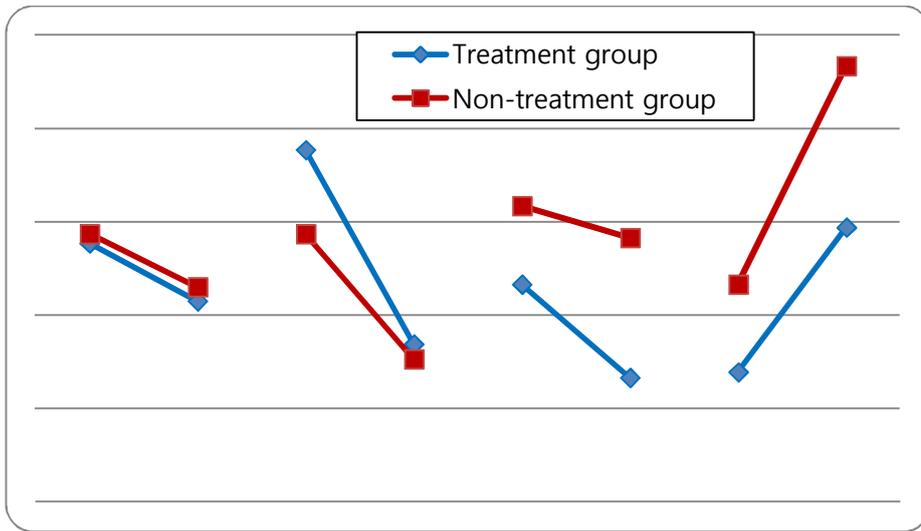


Figure 3.

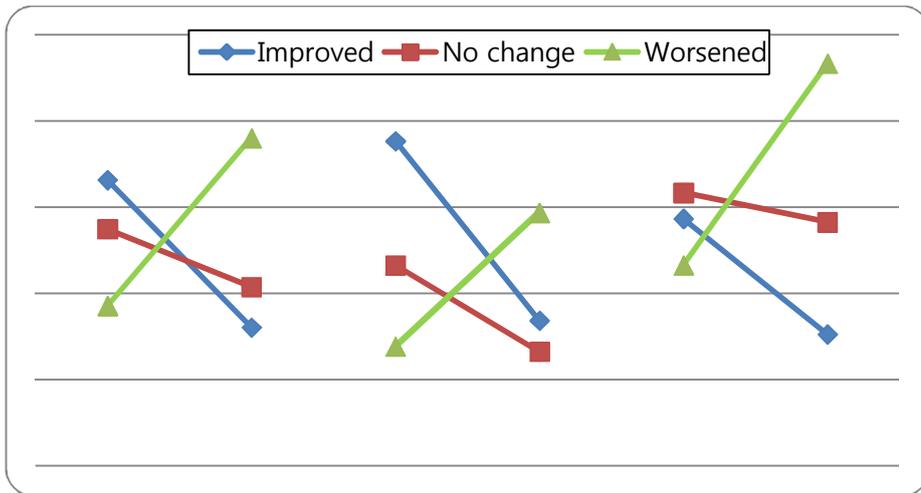


Figure 4.

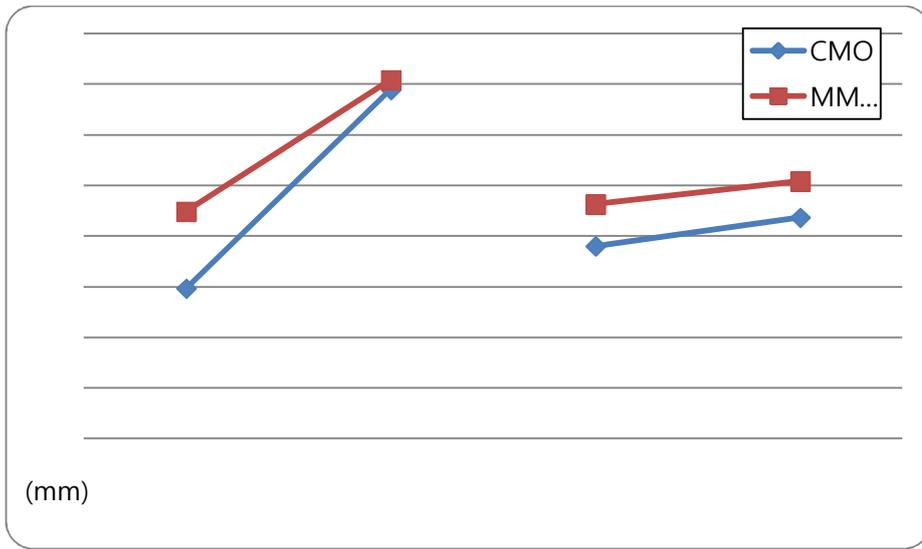


Figure 5.

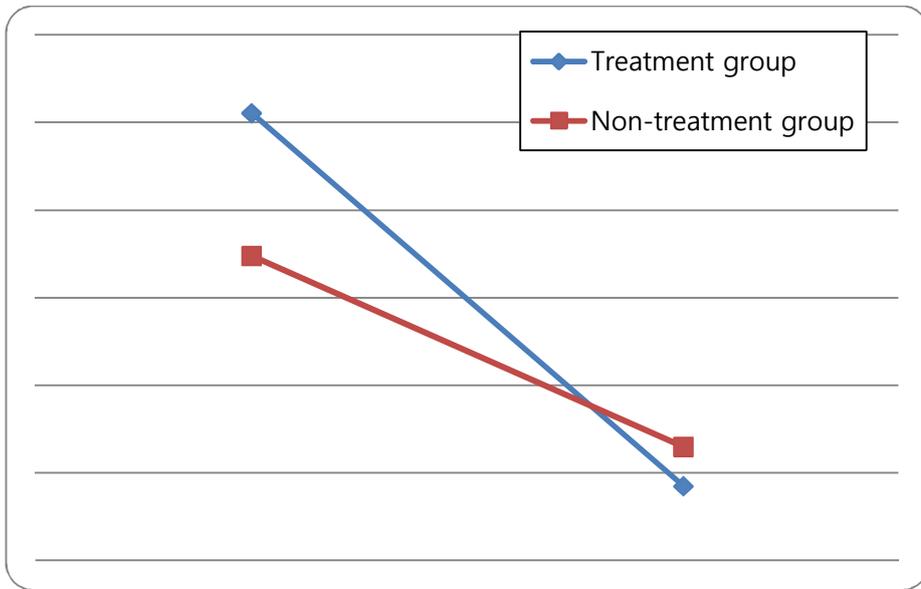


Figure 6.

젊은 연령의 측두하악관절 골관절염 환자의 골과괴 변화에 관한 전산화단층촬영을 이용한 치료군과 대조군의 종적 비교 연구

서울대학교 대학원 치의과학과 구강내과·진단학 전공
(지도교수 이 정 윤)

송 환 희

본 연구의 목적은 젊은 연령의 측두하악관절 골관절염(Temporomandibular joint osteoarthritis, TMJ OA) 환자들 가운데 전산화단층촬영(CT)에서 하악과두의 골과괴 변화가 확인되는 환자를 대상으로 하여 그 골변화의 양상과 예후를 치료군과 대조군을 비교하여 평가하는 것이다.

CT 영상에서 뚜렷한 골과괴가 확인되는 44명의 TMJ OA 환자의 CT 영상과 임상정보를 분석하였다. 치료군은 첫 내원시 CT 검사를 통해 TMJ OA로 진단되어 행동조절 교육, 물리치료 및 주기적인 내원을 통한 교합안정장치 치료를 받은 29명의 환자로 구성되었으며, 대조군은 첫 내원시 CT 검사를 통해 TMJ OA로 진단되었으나 질환에 대해 설명을 들은 후에 아무런 처치를 받지 않은 15명의 TMJ OA환자로 구성되었다. TMJ OA의 골변화 예후는 평균 1.5 ± 0.5 년 간격으로 촬영된 두 번의 CT

영상의 비교를 통해 분석하였고, 의무기록을 검토하여 임상적 특징과의 상관관계를 분석하였다. 첫 CT영상에서 골파괴가 확인된 70개의 관절(치료군 47관절, 대조군 23관절)을 종적인 CT영상 분석결과에 따라 향상, 변화없음 그리고 악화의 3개의 집단으로 분류하였다. 또한 전체적인 골파괴의 정도를 파악하기 위하여 관절표면을 인위적으로 9등분하여 침식 또는 subchondral bone cyst가 나타난 영역의 개수를 골파괴지수로 설정하여 비교 분석하였으며 결과는 다음과 같다.

1. 전체 관절 중 향상된 그룹의 관절수가 치료군과 대조군 모두에서 가장 높은 빈도(치료군 55.3%, 대조군 60.9%)로 관찰되었다.
2. 전체적인 골파괴지수의 변화는 치료여부나 시간의 변화에 따라 유의한 차이를 보이지 않았다.
3. 비록 통계적으로 유의하지 않더라도 전반적인 골파괴지수의 변화 양상은 대조군보다 치료군에서 우수한 것으로 관찰되었으며, 치료군의 초기 골파괴지수의 값은 예후가 향상된 집단에서 유의하게 높았고, 예후가 악화된 집단에서 초기 골파괴지수는 유의하게 낮았다.
4. 임상적 정보 분석 결과, 개구량의 범위는 대조군에 비해 치료군에서 유의하게 증가하였으며, 자가보고 통증의 유병률은 치료군에서 높았으나 통계적으로 유의하지는 않았다.

결론적으로 측두하악관절 골관절염의 하악과두의 골파괴는 질병 고유의 변화 양상으로 진행되는 것으로 보이며, 교합안정장치 치료를 포함하는 전통적인 치료법에는 덜 반응하는 것으로 나타났다. 그러나 행동조절 교육, 물리치료 및 주기적 내원을 통한 교합안정장치 치료 등은 골 변화 자체 뿐 아니라 임상증상 및 하악의 기능운동 개선에는

효과적이므로 측두하악관절 골관절염 환자의 치료에 있어 반드시 고려되어야 할 것이다.

주요어 : 측두하악관절, 골관절염, 전산화 단층촬영, 교합안정장치 치료

학 번 : 2010-23759